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Tubercular Consumption.—
Is it ever inherited?

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REPRINTED FROM PROCEEDINGS OF
The New York State Medical Association.
1885.





TUBERCULAR CONSUMPTION.—IS IT EVER INHERITED ?

By HENRY D. DIDAMA, M. D., of Onondaga County, N. Y.

Read November 17, 1885.

It will be assumed in this paper that tubercular consumption is infectious, and that for its causation the bacillus tuberculosis is a *sine quâ non*. Inoculations with inanimate matter never infect. The counterfeit tubercles produced by inhalations of powdered glass, or cheese, or dried mucus, are as sterile as a mule. Experimentation and discussion are of the greatest importance. But investigation should not be forever circular. To the devotees of science, as to the children of Israel of old, the injunction is: Go forward !

IS THE BACILLUS TUBERCULOSIS TRANSMITTED TO THE OVUM AT CONCEPTION, OR TO THE FÆTUS DURING GESTATION?—Cohnheim¹ in 1881 made this assertion: "That tuberculosis is hereditary is too well known to require to be more than stated." The virus "can be transmitted in the semen and in the ovum."

Klein, in a letter to the writer a month ago, says: "I think the assumption that the specific disease tuberculosis is transmitted through ovum or semen to the child is probably correct. If tuberculosis is a specific disease, caused by a specific organism and by nothing else—and that this is so there can, I think, be little reasonable doubt—then it follows that being a *tissue disease*, and not merely a disease of the blood, it is probably transmissible by semen or ovum. Is not syphilis a good example?"

Neisser,² of Breslau, admits the "theoretical *possibility* that

¹ Ziemssen's "Supplement," p. 345.

² Ziemssen's "Hand-Book of Skin Diseases," p. 278.

the bacilli migrate from the maternal into the foetal organism, or that a direct bacterial infection of the ovule or the semen occurs, in analogy with syphilis, relapsing fever, and the silk-worm disease." But he holds that, "in the great majority of cases, the infection is extra-uterine, and occurs after birth."

Ernst Ziegler, Professor of Pathology at Tübingen, in his "Pathological Anatomy," published this year, declares that "if the ovum be impregnated at a time when the generative organs are affected with a tubercular disease, or when the tubercle bacilli are distributed through the body in the secretions and the blood, it is *possible* that the bacilli may be transmitted to the foetus. It is furthermore *possible* that the foetus *in utero* may become infected from its tubercular mother. But no positive evidence in either of these two matters is at hand."

Admitting the possibility of this infection, still, if the ovum were actually inoculated by bacilli derived from the mother, or by those contained in infectious spermatozoa, or if, at a later period, contamination should take place through placental circulation, evidence should be furnished by abundant tubercles in the pre-natal and in the new-born offspring. For, as W. Watson Cheyne stated not very long ago: "It is hardly likely that the bacillus would remain quiescent during the nine months of foetal life, or during the years which often elapse before the development of what is supposed to be inherited tuberculosis."

HAS THE CLAIM THAT TUBERCULOSIS IS ANTENATAL BEEN ESTABLISHED?—Chaussier,¹ in 1812, claimed to have found tubercles in a foetus which died at birth. Husson reports that he found tubercles in the liver of a child eight days old, and in the lungs of a foetus still-born at the seventh month. Billiard met with evident tuberculous granulations in three of a great many infants examined. These observations were made seventy years ago, when histology was immature and unsatisfactory. Velpeau and Breschet never observed them in all their researches. Guizot looked for them in four hundred new-born children, but did not find a single example.

E. Wagner,² in 1876, asserted that tubercles had not then

¹ "Cyclopædia of Pract. Med.," p. 525.

² "General Pathology," p. 451.

with certainty been observed in the foetus; and that in tuberculosis, produced in animals by inoculation, tubercles had not been observed in the embryos or the newly born.

Epstein,¹ in 1879, stated that congenital tuberculosis is extremely rare, to say the least, and that for absolute development of it we must look for the subsequent introduction of the specific material.

Fränkel² declares that tuberculosis has been found once only at birth, and then by Virchow, who informed him personally of the case. Virchow has never himself published this observation. Ziegler affirms that "neither in a child at full term nor in a prematurely delivered human embryo has the presence of tubercles containing bacilli been positively established." One of the latest eminent authorities is Birch Hirschfeld,³ who, in the great work now running through the press, makes this important statement: "In contradistinction to syphilis, which often proves its hereditary transmissions by anatomical lesions in the foetus or new-born babe, *a well-authenticated case of tuberculosis at birth has never been seen.*" . . .

"From such experiences," he continues, "we must conclude that *the disease itself is not transmitted to the child or foetus*, but that those children have a disposition to the disease."

He further states that in those cases of supposed hereditary tuberculosis there is simply an infection resulting from cohabitation with tuberculous individuals.

In apparent contradiction of these statements are certain facts and deductions furnished by my friend Dr. P. M. Wise, of the Willard Asylum for the Insane. Extracts from his letter, sent to me before I commenced this paper, are subjoined. The facts presented seem to prove that the tuberculous pearl disease of cattle, in which bacilli are found, is transmitted to the offspring, and that, in a large number of cases, tubercles are found in countless numbers before birth, or immediately after birth.

¹ Ziemssen's "Supplement," p. 339.

² Gerhardt, "Handbuch der Kinder," vol. iii, p. 170.

³ "Pathological Anat.," vol. i, p. 175.

WILLARD'S ASYLUM FOR THE INSANE, May 28, 1885.

DEAR DOCTOR DIDAMA : Several years ago we had tuberculous consumption develop in our asylum herd of cattle of milch-cows to an alarming degree. During the winter of 1883-'84 the disease assumed an acute form. Dr. Blaine, an assistant physician of our household, made careful clinical observations, and a pathological study of all cases that died or were killed.

The etiology of a number of cases was traced to a diseased bull ; and, in the case of another bull that has since died from tuberculosis, his *seminal* wanderings can be traced through the country by the tuberculosis he has transmitted to his offspring.

The calves born during the past two years from consumptive cows are, almost without exception, diseased ; many of them have already succumbed. Twenty-nine calves killed under four months of age presented tubercular deposits in some of the viscera. One still-born calf was *saturated* with the tubercular deposit, and one *in utero* was found to have it. One calf, from an unquestionably healthy cow but from a diseased bull, was found, *post-mortem*, to have the characteristic disease. The congenital presence of the disease is proved in our experience over and over again. At the International Veterinary Congress at Brussels, in 1883, Lydton quoted more than a score of veterinary authors in favor of the heredity of tuberculosis. Gæring noticed that in bovine tuberculosis one hundred and twenty-three were infected by the dam and forty-three by the sire.

In answer to an objection that bovine and human tuberculosis are not identical, and hence comparisons are untrustworthy and without value, I desire to state that the presence of the *bacillus tuberculosis* is as essential to the diagnosis of bovine tubercle as it is in human tuberculosis.

Professor James Law, of Cornell University, examined our herd with care, and pronounced the disease tuberculosis. This is the *perl sucht* of the Germans. It invades primarily the lymphatics and serous membranes, and in this respect is distinctively different from the disease in man.

The pathological differences, however, are no wider than in several diseases common to man and animals ; and the presence of the specific organism would mark the two forms of disease as of a uniform nature. Dr. Creighton, at the International Medical Con-

gress, in London, maintained that the bovine tuberculosis, or *perl sucht*, could be communicated to children by the consumption of milk from the diseased cows.

Very sincerely yours,

P. M. WISE.

Cheyne, in his letter before quoted, says: "Johne¹ gives a case—the only undoubted one recorded—of congenital tuberculosis in a foetal calf of eight months. The cow was killed and found to have extensive tuberculosis of the lungs, but *not* of the placenta or uterus. The foetus had tubercles in lungs and liver containing tubercle bacilli." Ziegler, referring to this case, says that Johne believes the infection to have been through the placenta. The cases furnished by Dr. Wise are marked additions to the undoubted ones heretofore recorded.

Klein, in his "Micro-Organisms,"² asserts that the bacilli found in the pearl nodules are only about two thirds the size of those found in the tubercles of man. In his letter, already referred to, he states: "I still consider that the bacilli in human and bovine tuberculosis are not identical. Their morphological characters, their distribution, and their action on guinea-pigs and rabbits, prove this."

Cheyne, with nearly all German pathologists, holds a somewhat different opinion. He writes: "I do not agree with Klein that the bacilli in *perl sucht* and human tuberculosis are different. There is no ground whatever for such a view. Morphologically the bacilli are identical; the conditions under which they grow outside the body, and the naked-eye and microscopic appearances of the cultivation are the same; while their effect on animals after inoculation, inhalation, etc., is absolutely identical. Klein thinks that the *perl sucht* organisms differ in size from the others. But, supposing this to be the case—and I am not at all sure that it is so—this is not sufficient to separate them as distinct organisms, when they agree in all the other and more important characteristics. Slight differences in size may depend on the mode of staining, stage of growth of the organism, soil, etc."

¹ "Fortschritte der Med.," No. 7, vol. iii, 1885.

² "Micro-Organisms and Disease," p. 126.

Further investigations seem to be needed to reconcile the views of these eminent observers.

Dr. Creighton, it will be noticed, does not claim that human tuberculosis was ever produced in children by the milk of a diseased cow, but merely that pearl nodules have been ; while Virchow makes the important statement that no human being ever got a pearl tumor from eating the flesh of an animal which had that disease.

If the bacilli in the bovine and in the human tuberculosis be not identical, there can be no justifiable inference that seminal transmission takes place in one case because it may take place in the other. But, granting that the bacilli are identical in the two complaints, there is no denial that the bovine disease is unlike the human variety in its location, its development, and its appearance. Why may not this dissimilarity be such that one shall be transmitted through the ovules and the other not? How otherwise account for the frequent existence of tubercles in new-born calves, and their entire absence in new-born babes? That certain diseases are transmitted from parents to unborn offspring may be admitted; syphilis and bovine tuberculosis may be examples; but this does not affect the allegation that human tuberculosis is not so transmitted—an allegation supported by a great multitude of competent observations.

At this point it may be assumed as incalculably more than probable that the specific disease tuberculosis is not inherited by the child.

DOES IT DERIVE A CERTAIN MYSTERIOUS DYSCRASIA OR TENDENCY TO THE DISEASE FROM ITS CONSUMPTIVE PARENTS?—Handed down through many centuries has been the doctrine that a tuberculous diathesis is inherited. Sixty years ago Armstrong¹ asserted that phthisis occurs only in the strumous temperament, and that it remained to be proved whether tubercles are ever found in the lungs without a hereditary predisposition to them.

Sir James Clark,² in 1845, declared that he regarded the

¹ "Fevers, Consumption," etc., p. 85.

² "Cyclop. of Pract. Med.," vol. iv, p. 535.

transmission of the tuberculous constitution from parent to child as one of the best established points in the ætiology of the disease.

Sir Thomas Watson avowed his belief in a hereditary diathesis.

Flint,¹ in 1873, cited, as proof that a tuberculous diathesis is in certain cases congenital and inherited, many instances where, the mother, or both parents, having died of consumption, all the children—five, seven, or even nine, in some families—died of the same disease. He is now a firm believer in the bacillary origin of phthisis, with or without inherited tendency.

Ruehle,² in 1875, claimed that no physician who makes an unbiased examination can reject the concurrent testimony of all times that consumption is hereditary from a constitutional taint whose nature is unknown.

Bristowe,³ in 1876, believed that a hereditary taint is strongly shown; that parents free from tubercle may transmit a latent taint which shall reveal itself as phthisis in the children. But he admits that the tendency to consumption which some children seem to possess may come from parents whose health has been impaired from non-tuberculous diseases.

Roberts, in 1884, declared that there can be no doubt as to the inherited tendency to phthisis, and Loomis traces this tendency definitely to the father in some cases and in others to the mother.

Tendency is a continual inclination. But that a continual inclination to consumption is inherited, any more than is the specific disease, may well be doubted.

Nearly twenty years ago Niemeyer announced that a consumptive parent, or one broken down by age, disease, debauchery, or vices, transmits a feeble vitality, an impaired resisting power.

In the light of recent discoveries we can see that the so-called diathesis or tendency is nothing more than cellular im-

¹ "Practice Med.," p. 295.

² Ziemssen's "Cyclop.," vol. v, p. 482.

³ "Practice of Med."

puissance inherited from parents broken down with any disease, or acquired from bad air, poor food, insufficient sunlight, unhealthy occupations, or other debilitating influences. The feeble and fragile cells furnish the bacilli a suitable soil for development and offer an impotent resistance.

Forty years ago Walshe carefully analyzed and recorded the family history of one hundred and two phthisical patients admitted into the Brompton Hospital for Consumption. Twenty-six per cent only of these had a tuberculous parent. Considering the prevalence of consumption, it is safe to assert that twenty-six per cent of all patients in hospitals—those afflicted with typhoid fever, kidney disease, and liver complaint—have a consumptive parentage. Does this prove that a tendency to these diseases was inherited from the tuberculous ancestors? Seventy-four per cent of the consumptive patients did not have tuberculous ancestors. Will it be pretended that these *non-tuberculous* parents transmitted a taint which they did not possess? A *latent* taint is a pitiful assumption.

It is well-known that the greatest care is taken by life-insurance companies to reject not only those applicants who have suspicious pulmonary symptoms, but those also who are connected by ties of consanguinity with consumptives. And what is the outcome of all this care?

Dr. Shepherd, of the Connecticut Mutual, informs me that, of their first 5,000 deaths, nearly 20 per cent were from tubercular disease; while of the next 5,000—partly owing to the selection of medical examiners competent to detect incipient disease—about 16 per cent died from that cause.

Dr. Lambert, of the Equitable, writes that, of their first 3,000 deaths, 21 per cent were from tubercular disease. Their later experience has been more favorable. While the doctor thinks, in common with most medical officers of insurance companies, that there is a family tendency, he states as his impression that "men who are poorly nourished, men whose weight is not in proportion to their height, do die of consumption much more rapidly than those who have a better nourishment, irre-

spective of the fact of consumption appearing as a factor in their family history."

Meech, of Chicago, quoted by Bell in his recent work on climatology, gives reports from twenty-seven insurance companies. Of 37,000 deaths, 20 per cent were from consumption.

These statistics and those from the Brompton Hospital confirm daily observation that a large majority of consumptive cases come from parents who are not phthisical, and who, consequently, could not transmit a dyscrasia, diathesis, tendency or disposition.

A mysterious tendency, which is always fruitless without bacilli, is unnecessary to account for any of the facts of phthisis, and there is no good reason for believing that it exists.

But if a weak constitution only, and not a specific predisposition, be transmitted, why do the children of tuberculous parents have consumption more uniformly than the children of the old man, the drunkard, or the syphilitic?

The answer is obvious. The former are more exposed to the bacillic infection. They live in an atmosphere contaminated with tuberculous poison. Bacilli are everywhere present in cities and crowded apartments; but they are specially abundant in the dwellings of the consumptive. The weakly babe, sleeping in the arms of its diseased mother, breathing her infecting breath, nursing impoverished if not poisonous milk from her unhealthy breasts; why should it not catch the phthisis sooner than its feeble neighbor, who inherits just the same good-for-nothing constitution, but has no special and constant exposure to infection? And, if it escapes in babyhood, why should it not yield to the assaults of the multitudinous bacilli in after years?

Epstein states that the children of tuberculous parentage do well when given to healthy nurses and kept away from tuberculous people; and that, if death then takes place, tubercles are never found at the autopsy. On the other hand, children suckled by tuberculous mothers become tuberculous soon after the development of the pulmonary affection in the mother.

In the remarkable cases recorded by Flint, where both parents and nine children died of consumption, the inference which he to-day must draw is, not that the disease was inherited, but that it was caught. The inherited weak cells were powerless against such an army of bacillie invaders.

THE ÆTIOLOGICAL CONCLUSIONS OF THIS PAPER ARE:

1. Tuberculous phthisis is **not** inherited.

2. Neither is a special *tendency* to the disease transmitted. In *fragilitas ossium*, the great brittleness of the bones is not spoken of as a *tendency* to fracture; liability is the better expression. An inherited tendency to catch smallpox would be a double absurdity.

3. Many conditions—as poor and insufficient food, damp and impure air, stinted sunlight, low altitudes, certain occupations—favor the taking and development of the disease.

4. Two conditions are almost indispensable: *abundance* of bacilli, and an inviting asylum for them, furnished by an inherited or acquired cellular *vincibility*.

These conditions predominate in the abodes of consumptives.

THE THERAPEUTIC SUGGESTIONS ARE:

1. The new-born babe of a tuberculous mother should be committed at once to the tender mercies of a healthy wet-nurse, who should occupy well-aired and sunshiny rooms, at a distance from the apartments of every one having a chronic pulmonary affection.

While this isolation from unhealthy surroundings affords no infallible protection against subsequent contamination—since the feeble constitution may still remain—it gives time to fortify the health and so lessen, if not eradicate, the liability to infection.

2. If a syphilitic taint in either parent be known or suspected, prolonged specific treatment of the infant should be instituted; not alone to remove the existing inherited disease, but to strengthen against tuberculosis, which is liable and likely to be superadded. Continued efforts to overcome inherited or acquired weakness may prevent bacillary infection or hinder its development.

